The American Journal of Surgery (2011) 202, 404–408

The American Journal of Surgery®

Clinical Science

Hypotension is 100 mm Hg on the battlefield

Brian J. Eastridge, M.D.*, Jose Salinas, Ph.D., Charles E. Wade, Ph.D., Lorne H. Blackbourne, M.D.

Trauma and Surgical Critical Care, US Army Institute for Surgical Research, 3400 Rawley E. Chambers Ave., MCMR-SRJ, Fort Sam Houston, TX 78234–6315, USA

KEYWORDS:
Hypotension;
Shock;
Trauma;
Combat;
Base deficit;
Systolic blood pressure;
Mortality

Abstract

BACKGROUND: Historically, emergency physicians and trauma surgeons have referred to a systolic blood pressure (SBP) of 90 mm Hg as hypotension. Recent evidence from the civilian trauma literature suggests that 110 mm Hg may be more appropriate based on associated acidosis and outcome measures. In this analysis, we sought to determine the relationship between SBP, hypoperfusion, and mortality in the combat casualty.

METHODS: A total of 7,180 US military combat casualties from the Joint Theater Trauma Registry from 2002 to 2009 were analyzed with respect to admission SBP, base deficit, and mortality. Base deficit, as a measure of hypoperfusion, and mortality were plotted against 10-mm Hg increments in admission SBP.

RESULTS: By plotting SBP, baseline mortality was less than 2% down to a level of 101 to 110 mm Hg, at which point the slope of the curve increased dramatically to a mortality rate of 45.1% in casualties with an SBP of 60 mm Hg or less but more than 0 mm Hg. A presenting SBP of 0 mm Hg was associated with 100% mortality. The data also established a similar effect for base deficit with a sharp increase in the rate of acidosis, which became manifest at an SBP in the range of 90 to 100 mm Hg.

CONCLUSIONS: This analysis shows that an SBP of 100 mm Hg or less may be a better and more clinically relevant definition of hypotension and impending hypoperfusion in the combat casualty. One utility of this analysis may be the more expeditious identification of battlefield casualties in need of life-saving interventions such as the need for blood or surgical intervention.

The most substantial clinical problem facing physicians on the battlefield is the identification of combat casualties at risk for continued hemorrhage and the potential for subsequent mortality.[1,2] Fundamental to this issue is the knowledge that diagnostic techniques are often rudimentary in the tactical environment, leading physicians to rely heavily on their innate diagnostic skill along with simple physiological measures to make complex clinical decisions. Prior studies from the battlefield have noted a strong correlation between a combination of several admission anatomic and physiological measures and the need for massive transfusion.[3] However, the relevance of systolic blood pressure (SBP) alone as a predictor of outcome or necessity for intervention after battlefield injury has not been rigorously studied.

Because of its simplicity, blood pressure continues to serve as a persistent indicator for early diagnostic and therapeutic decision making after injury. Current standards typically use SBP as a triage tool and as one of the measured

The opinions or assertions expressed herein are the views of the authors and are not to be construed as official or as reflecting the views of the US Army or the US Department of Defense.

The authors have no financial or proprietary interest in the subject matter and no other identifiable conflict of interest.

* Corresponding author. Tel.: +1-210-916-7104; fax: +1-210-916-1602.
E-mail address: brian.eastridge@amedd.army.mil

Manuscript received July 29, 2010; revised manuscript October 1, 2010

0002-9610/$ - see front matter Published by Elsevier Inc.
doi:10.1016/j.amjsurg.2010.10.012

Report Documentation Page

Form Approved
OMB No. 0704-0188

1. REPORT DATE	2. REPORT TYPE	3. DATES COVERED
01 OCT 2011	**N/A**	**-**

4. TITLE AND SUBTITLE	5a. CONTRACT NUMBER
Hypotension is 100 mm Hg on the battlefield	5b. GRANT NUMBER
	5c. PROGRAM ELEMENT NUMBER
6. AUTHOR(S)	5d. PROJECT NUMBER
Eastridge B. J., Salinas J., Wade C. E., Blackbourne L. H.,	5e. TASK NUMBER
	5f. WORK UNIT NUMBER
7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES) **United States Army Institute of Surgical Research, JBSA Fort Sam Houston, TX**	8. PERFORMING ORGANIZATION REPORT NUMBER
9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)	10. SPONSOR/MONITOR'S ACRONYM(S)
	11. SPONSOR/MONITOR'S REPORT NUMBER(S)

12. DISTRIBUTION/AVAILABILITY STATEMENT
Approved for public release, distribution unlimited

13. SUPPLEMENTARY NOTES

14. ABSTRACT

15. SUBJECT TERMS

16. SECURITY CLASSIFICATION OF:			17. LIMITATION OF ABSTRACT	18. NUMBER OF PAGES	19a. NAME OF RESPONSIBLE PERSON
a. REPORT	b. ABSTRACT	c. THIS PAGE			
unclassified	**unclassified**	**unclassified**	**UU**	**5**	

values to determine patient injury severity. Furthermore, the association between hypotension, hypoperfusion, and poor outcomes in trauma patients are well described.[4,5] Although hypotension in trauma patients often is defined as an SBP of 90 mm Hg or less, little data exist to support the dogmatic adherence to this arbitrary value. In our previous study of civilian trauma patients using the National Trauma Data Bank (NTDB), we were able to corroborate the predictive value of admission metabolic acidosis on patient survival. We also were able to show that an SBP of 110 mm Hg after injury was associated with physiological and adverse outcome consequences. The value of 110 mm Hg appeared to be the uniform inflexion point at which mortality, metabolic acidosis, complications, length of stay, intensive care unit days, and ventilator days all increased. The implication of our result suggested the insidious evolution of shock, with attendant increases in injury-associated complications, which was independent of both the age and sex of the patient.[6]

The premise of the current study is that the practice of prehospital and initial battlefield medical treatment facility triage of the combat casualty may be improved significantly by providing a more liberal definition of hypotension, which in turn may provide an earlier and better indicator of blood volume loss and impending circulatory collapse. Therefore, we hypothesized that physiological hypoperfusion and mortality outcomes classically associated with hypotension were manifest at SBPs greater than 90 mm Hg.

Materials and Methods

A retrospective review of patient records from the Joint Theater Trauma Registry (JTTR) was analyzed for this study. Records consisted of patient trauma registry data collected from military medical treatment facilities within Iraq and Afghanistan. A total of 7,180 patient records from the JTTR from 2002 to 2009 with emergency department SBP, base deficit, and mortality data were analyzed. A total of 16,476 individual casualty records were in the JTTR at the time of this analysis. Patients with moderate to severe traumatic brain injury (Head Abbreviated Injury Score ≥ 3) were excluded from the analysis to limit confounding by neurologic causes of hypotension. Patients also were excluded for lack of any requisite data element. Analysis was performed on SBP relative to hypoperfusion manifest as an increase of base deficit and mortality rate. Summary data were plotted and filtered using window average filters to decimate peaks in the resulting dataset. Inflection points in the filtered dataset were determined by calculating the data slope for each point in the filtered data set. Average, median, and/or average line slope estimation filters were used to generate a filtered slope set for each variable. All filters were executed using a moving window across values in the resulting dataset to generate the filtered results. The point of inflection in the final data set was determined by the point at which the filtered slope values maintain a positive slope and do not cross the 0 line.

Results

The composite dataset showed a baseline mortality rate of less than 1%. Figure 1 shows the plot of SBP compared with mortality for the dataset. These data were notable for an initial inflection point at the decile of 101 to 110 mm Hg. The slope of the line at SBP below this point had an initial increase of approximately 4% in mortality for the next decile in SBP followed by a 10% increase in mortality for every decrease of 10 mm Hg, with a maximum of 33% mortality at an SBP of 61 to 70 mm Hg. Patients with measurable SBP less than 60 mm Hg had a mortality rate of 45%. No patient without a discernable blood pressure survived. In the analyzed group

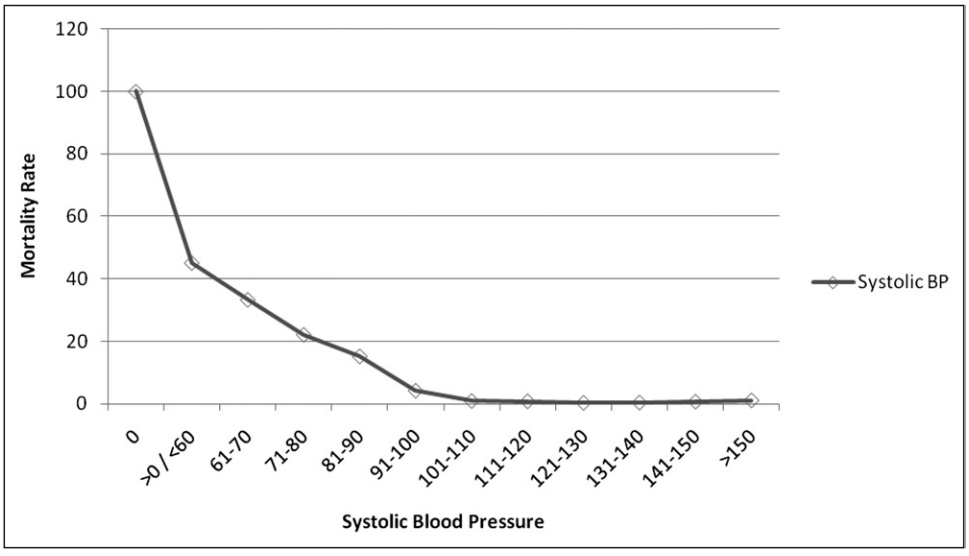

Figure 1 Systolic BP and mortality.

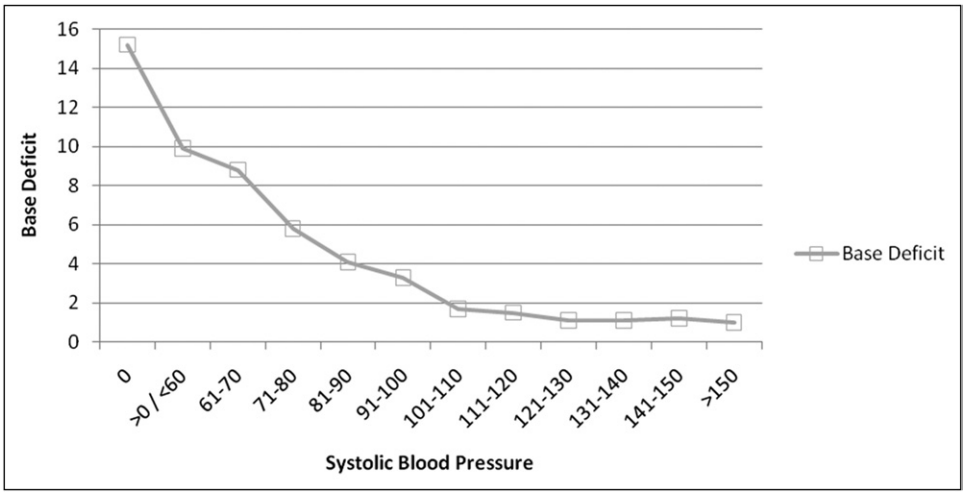

Figure 2 Systolic BP and base deficit.

of casualties, 486 (6.8%) presented with a SBP of 100 mm Hg or less; whereas 6,694 casualties presented with an SBP of greater than 100 mm Hg.

Figure 2 shows SBP compared with base deficit. At 101 to 110 mm Hg the slope of the curve began a sustained and significant increase. The resultant composite perfusion deficit, as indicated by a change in the slope of base deficit curve, increased steadily above baseline of 1.5.

Comments

Current trauma triage relies on abnormal physiological criteria to determine the patient's mode of transport, priority of treatment, destination for treatment, injury severity, mortality, and need for possible life-saving interventions. However, the physiological portion of most of these existing triage tools is based on the presence of abnormal vital signs in the patient, particularly an SBP of 90 mm Hg or less. Common vital signs are used because these measurements usually are readily obtainable at the site of injury and therefore may provide a snapshot of patient stability. However, such an assumption is problematic because the physiology of the trauma patient suffering from severe hemorrhage is often dynamic and may not reflect the true degree of hypoperfusion present owing to normal physiological compensatory mechanisms. Furthermore, previous literature has shown that significant hypoperfusion occurs in hypovolemic laboratory experiments as well as in blunt and penetrating trauma patients despite normal, standard vital signs, especially in young healthy patients.[7,8] Compensatory mechanisms allow significant reductions in central circulating blood volume, stroke volume, and cardiac output to occur well before changes in arterial blood pressure. Such physiological compensations can thus mask the true nature and severity of many traumatic injuries, leading to underappreciation of the severity of injury, under-triage, and an increased mortality rate. In fact, an SBP of 90 mm Hg signifies impending cardiovascular collapse and underscores the necessity for improved indicators to improve triage and initiate early intervention strategies to improve postinjury mortality.[9] Although an initial SBP of 90 mm Hg or less has been shown previously to provide higher sensitivity and better specificity for prediction of mortality and outcome than most traditional vital signs (respiratory rate, heart rate, and so forth),[10–14] more sensitive markers of acute hypoperfusion are needed if hemorrhage and circulatory shock are to be recognized in its early stages when life-saving interventions are most likely to be successful.

Our analysis of the combat injury data suggest that an SBP of 100 mm Hg or less is associated with the initiation of physiological perturbations and outcome consequences. This result is entirely consistent with our previous work on the civilian population. We postulate that the lower SBP associated with acidosis and mortality in the current study is reflective of the attributes of the military population who are generally younger and healthier and have greater physiological reserve than the composite civilian cohort. In fact, the cohort of combat casualties studied was largely from the combat arms community. Because of the nature of being constantly in harm's way on combat missions, these warriors are generally supremely conditioned, much akin to a world-class athlete. To illustrate a similar point from our prior analysis, the SBP of the composite group showed that the blood pressure inflection indicative of hypoperfusion and mortality was 110 mm Hg. However, when stratified by age, patients who were age 43 and older had hypotension defined as 117 mm Hg, whereas patients younger than age 43 showed hypotension at 108 mm Hg.[6]

To more comprehensively sustain our contention relative to the importance of liberally defining hypotension, there have been a few contemporary series in the civilian trauma environment that have highlighted the lack of clinical substance to support 90 mm Hg as a physiologic definition of shock.[15–19] In a study of 19,409 injured patients, Arbabi et al[16] stratified patients by 30-mm Hg increments and showed

a baseline mortality rate of approximately 5% and a similar incremental increase in mortality with decreasing SBP. In a hallmark study, Franklin et al[17] evaluated the impact of prehospital hypotension in a cohort of 299 injured patients with at least one documented episode of field or emergency department hypotension (SBP, <90 mm Hg), as a valid indicator of trauma team activation. In this series, patients with field and emergency department hypotension had an emergent surgical indication in 79% of patients with a mortality of 44%. Hypotension was presented as an important predictor of outcome, but not defined further. In 2,071 injured patients requiring exploratory laparotomy with gastric, small-bowel, and/or diaphragm injuries, Edelman et al[19] showed a baseline mortality rate of patients with an SBP of 110 mm Hg or greater that was less than 1%; however, it increased to 5% in the population with an SBP between 90 and 110 mm Hg. Concomitant with this increase in mortality were significant increases in length of stay and infection.

Our study had notable limitations. The data used for our analyses were derived from the JTTR. By its nature, the JTTR has a number of inherent limitations, including its basis as a composite registry of numerous military medical treatment facilities collected across the continuum of casualty evacuation under challenging environmental and situational circumstances. Any potential deficiencies in these contributing entities are reflected in the overall database. Other potential limitations of the JTTR include data validation because of the size of the database, output issues associated with nonsystematic sampling, and selection and information bias. Specifically, patients also were excluded from our analysis for lack of any requisite data element. Therefore, only 7,180 of 16,476 (43.6%) casualties in the JTTR were available for analysis. The most frequently missing data elements causing exclusion was base deficit.

As in our previous study, we also must acknowledge that the current investigative effort has several other important limitations not intrinsically related to the database. First, this analysis was based on the first SBP in the emergency department and thus the same threshold may not be applicable to the prehospital setting. Similar to the NTDB, the JTTR dataset is restricted by the lack of prehospital times and resuscitative fluid volumes. Another potential limitation of our study was blood pressure measurement itself. Many emergency departments in deployed medical treatment facilities use automated blood pressure recording devices for SBP measurement. Previous investigations have shown a propensity for automated devices to overestimate the true manual blood pressure by approximately 10 mm Hg.[20,21]

Conclusions

Our current analysis illustrates an SBP of 100 mm Hg or less to be a more clinically relevant definition of hypotension and hypoperfusion than 90 mm Hg in the combat-wounded. We emphasize that this value cannot be considered in isolation owing to the overall lack of predictive value of the SBP measure. Furthermore, this should not be interpreted as an end point of resuscitation, but rather a more liberal set point for a heightened index of suspicion that the battlefield casualty may require more expeditious or comprehensive diagnostics to exclude significant injury or hasten therapy, such as blood transfusion or surgical intervention. This analysis further serves to establish that the development of new approaches for the prediction and early detection of shock in military casualty patients continues to be a priority.

References

1. Holcomb JB, McMullin NR, Pearse L, et al. Causes of death in US special operations forces in the global war on terrorism, 2001–2004. Ann Surg 2007;245:986–91.
2. Kelly JF, Ritenour AE, McLaughlin DF, et al. Injury severity and causes of death from Operation Iraqi Freedom and Operation Enduring Freedom: 2003–2004 versus 2006. J Trauma 2008;64:S21–6.
3. McLaughlin DF, Niles SE, Salinas J, et al. A predictive model for massive transfusion in combat casualty patients. J Trauma 2008;64: S57–63.
4. Husain FA, Martin MJ, Mullenix PS, et al. Serum lactate and base deficit as predictors of mortality and morbidity. Am J Surg 2003;185: 485–91.
5. Davis JW, Parks SN, Kaups KL, et al. Admission base deficit predicts transfusion requirements and risk of complications. J Trauma 1996; 41:769–74.
6. Eastridge BJ, Salinas J, McManus JG, et al. Hypotension begins at 110 mm Hg: redefining "hypotension" with data. J Trauma 2007;63:291–9.
7. Scalea TM, Holman M, Fuortes M, et al. Central venous blood oxygen saturation: an early, accurate measurement of volume during hemorrhage. J Trauma 1988;28:725–32.
8. Falk JL, Rackow E, Astiz MA, et al. Fluid resuscitation in shock. J Cadiothorac Anesth 1988;2:33–8.
9. Convertino VA, Cooke W, Holcomb JB. Arterial pulse pressure and its association with reduced stroke volume during progressive central hypovolemia. J Trauma 2006;61:629–34.
10. Abou-Khalil B, Scalea TM, Trooskin SZ, et al. Hemodynamic responses to shock in young trauma patients: need for invasive monitoring. Crit Care Med 1994;22:633–9.
11. Esposito TJ, Offner PJ, Jurkovich GJ, et al. Do prehospital trauma center triage criteria identify major trauma victims? Arch Surg 1995; 130:171–6.
12. Guzzo JL, Bochicchio GV, Napolitano LM, et al. Prediction of outcomes in trauma: anatomic or physiologic parameters? J Am Coll Surg 2005;201:891–7.
13. Siegel JH, Rivkind AI, Dalal S, et al. Early physiologic predictors of injury severity and death in blunt multiple trauma. Arch Surg 1990; 125:498–508.
14. Victorino GP, Battistella FD, Wisner DH. Does tachycardia correlate with hypotension after trauma? J Am Coll Surg 2003;196:679–84.
15. Holcomb JB, Salinas J, McManus JM, et al. Manual vital signs reliably predict need for life-saving interventions in trauma patients. J Trauma 2005;59:821–9.
16. Arbabi S, Jurkovich GJ, Wahl WL, et al. A comparison of prehospital and hospital data in trauma patients. J Trauma 2004;56:1029–32.
17. Franklin GA, Boaz PW, Spain DA, et al. Prehospital hypotension as a valid indicator of trauma team activation. J Trauma 2000;48:1034–9.

18. Luna GK, Eddy AC, Copass M. The sensitivity of vital signs in identifying major thoracoabdominal hemorrhage. Am J Surg 1989; 157:512–5.
19. Edelman DA, White MT, Tyburski JG, et al. Post-traumatic hypotension: should systolic blood pressure of 90–109 mm Hg be included? Shock 2007;27:134–8.
20. Davis JW, Davis IC, Bennink LD, et al. Are automated blood pressure measurements accurate in trauma patients? J Trauma 2003;55:860–3.
21. Wo CC, Shoemaker WC, Appel PL, et al. Unreliability of blood pressure and heart rate to evaluate cardiac output in emergency resuscitation and critical illness. Crit Care Med 1993;21:218–23.